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Response to Treatment of Hyperuricemia in Essential Hypertension

Philip B. Mellen, Anthony J. Bleyer and David M. Herrington

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Letter to the Editor

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Epidemiologic evidence linking serum uric acid and incident hypertension is rapidly proliferating, and animal models of hyperuricemia have helped elucidate possible mechanisms for this relationship.[1] Furthermore, secondary analyses of clinical trial data suggest that the relationship between hypertension treatment and clinical outcomes is related to serum uric acid levels.[2,3] The logical next step is to evaluate whether targeting serum uric acid prevents hypertension or hypertension-associated end-organ damage. We agree with Trachtman[4] that well-designed, randomized, controlled trials should evaluate the efficacy of uric acid–lowering therapy in the prevention of hypertension.

We also note that the failure of allopurinol to prevent hypertension in spontaneously hypertensive rats does not necessarily contradict the hypothesis that uric acid plays a causal role in human hypertension. As the study by Trachtman et al[5] noted, serum uric acid levels do not differ between the spontaneously hypertensive rat and the normotensive Wistar–Kyoto strain. Like most mammals (and in contrast to humans), rats possess the hepatic enzyme uricase, which facilitates the rapid degradation of uric acid. Hyperuricemic rat models of hypertension have used oxonic acid to inhibit uricase, yielding modest elevations in serum uric acid more consistent with those seen in humans.[1]

Forthcoming studies will address whether uric acid is a viable target for the pharmacological prevention of hypertension in humans. If the results are positive, they will require a careful scrutiny of adverse outcomes, given the potentially broad application of this strategy for the prevention of hypertension.

Disclosures

None.

Philip B. Mellen
Department of Internal Medicine
Section of General Medicine
Wake Forest University Health Sciences
Winston-Salem, NC

Anthony J. Bleyer
Department of Internal Medicine
Section of Nephrology
Wake Forest University Health Sciences
Winston-Salem, NC

David M. Herrington
Department of Internal Medicine
Section of Cardiology
Wake Forest University Health Sciences
Winston-Salem, NC

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